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## Pancreatic injury in COVID-19: pathogenesis and challenges

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*Dear Editor,*

Fernandes et al. reported a case of acute pancreatitis (AP) in a patient with SARS-CoV-2 (1). Although the authors make a detailed analysis of the possible causes of the AP (direct cytopathic effect of SARS-CoV-2 replication, severe systemic inflammatory response), they make no mention of the possible thrombotic etiopathogenesis of the AP.

Since the beginning of the SARS-CoV-2 pandemic, one notable feature of patients admitted with the disease has been the frequency of thrombotic and ischemic phenomena (myocardial infarction, stroke, ischemia of the lower extremities, intestinal ischemia), and even the development of disseminated intravascular coagulation (2). SARS-Cov-2 infection is associated with a severe immune response and the release of proinflammatory cytokines (systemic cytokine storm) and procoagulant (3).

The gastrointestinal tract is the largest immune organ and the enterocytes, together with the pancreatic islets and the endothelial cells found there, express abundant angiotensin-converting enzyme 2-related carboxypeptidase (ACE-2). Thus facilitating the entry of SARS-CoV-2.

Tang H. reported that 71.6 % of patients who died of COVID-19 developed disseminated intravascular coagulation, as compared to 0.6 % of survivors (2). Secondly, in patients with severe COVID-19, an increase in von Willebrand factor has been reported as a result of endothelial damage and the development of thrombotic phenomena (4).

In our center, we have treated three patients with severe ischemia of the colon, one of them associated with pancreatic necrosis (5). Apart from the mechanisms reported by Fernandes et al. (1), this thromboembolic pathogenesis should also be taken into account in patients with severe COVID-19 and prophylaxis with low molecular weight heparin should be implemented (2).

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